Foreign Animal Disease Report

United States Department of Agriculture
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In This Issue

Current Events

Emergency Field Operations

During the period October 1, 1987, through May 31, 1988, 156 investigations of suspected foreign animal diseases were conducted by Veterinary Services (VS) and State-employed veterinarians to determine the possibility that an exotic disease may have been introduced into the United States. No exotic diseases were found in 94 investigations for suspected vesicular disease, 26 for suspected hog cholera or African swine fever, 1 for equine encephalitis, 3 for suspected rinderpest or mucosal disease, 6 for avian influenza, 16 for Newcastle disease, and 4 for suspected but unspecified diseases or conditions.

Exotic Newcastle disease. Exotic velogenic viscerotropic Newcastle disease (VVND) was diagnosed in pet birds in the following six instances, during the period October 1, 1987, through May 31, 1988.

On April 20, 1988, the National Veterinary Services Laboratories (NVSL) at Ames, Iowa, confirmed VVND in specimens from a 3-week-old spectacled Amazon parrot. The parrot had severe central nervous system (CNS) signs when presented to a veterinarian in Detroit, Michigan. A Newcastle virus isolated from the bird at the Michigan Animal Health Laboratory, Lansing, Michigan, had been forwarded to NVSL for further identification.

On April 23, 1988, exotic Newcastle disease was confirmed in a young Yellow-naped parrot which had been presented to a veterinarian in Las Vegas, Nevada. Since the bird was showing CNS signs, the veterinarian contacted a VS Veterinary Medical Officer who then submitted the bird to NVSL. The bird was one of eight young Yellow-napes which reportedly had been purchased at a bird "swap meet" (informal retail sales event) in San Diego, California, by a Las Vegas resident who then sold two of them to the person on whose premises exotic Newcastle disease was found. Also in April, exotic Newcastle disease was confirmed in a young Yellow-nape parrot which reportedly was purchased from a vendor in Los Angeles, California.

In May, the California State Diagnostic Laboratory identified VVND virus from a conure owned by a person in Duarte, California. The bird reportedly was purchased at a swap meet. Later that month, a young spectacled Amazon parrot with CNS signs was presented to a veterinarian in Niles, Illinois. Specimens were submitted to NVSL where VVND was confirmed. The sick bird and another one that had died earlier reportedly were purchased at a street corner in Chicago.

Elsewhere in May, two young half-moon conures were presented to a veterinarian in El Paso, Texas, who then submitted one of them to the Texas A&M University Veterinary Diagnostic Laboratory. The submitted bird had died following the onset of CNS signs. VVND virus was identified on May 27, 1988, in specimens the Texas laboratory forwarded to NVSL. The conures reportedly were purchased in Mexico and apparently were brought into the United States without having met Federal import requirements.

Intensive investigations were conducted to locate any other infected and exposed birds. No additional cases have been found, and there is no indication that infected birds have been in pet bird commercial channels. All infected birds were humanely destroyed, and affected premises were cleaned and disinfected. Evidence to date indicates that these outbreaks involved birds which were brought into the United States contrary to quarantine rules and other requirements for importing birds.

Training. A training course for veterinarians in the U.S. Army Veterinary Corps who provide emergency support to Veterinary Services (VS) was held in Hyattsville, Maryland, April 5-8, 1988. Foreign Animal Disease Diagnosticians from the Western and Central VS Regions of the United States who had not attended a training course on foreign animal diseases during the past 4 years attended a Foreign Animal Disease Seminar May 24-26, at Tulsa, Oklahoma. A foreign animal disease training course was held May 2-20, 1988, with segments at the National Veterinary Service Laboratories (NVSL) at Ames, Iowa; Foreign Animal Disease Diagnostic Laboratory and Plum Island Animal Disease Center, Greenport, Long Island, New York; and VS headquarters, Hyattsville, Maryland. Trainees included veterinarians from the Animal and Plant Health Inspection Service (APHIS), State of California, and one from Australia.

READI System. A Recorded Emergency Animal Disease Information (READI) system test exercise was conducted in Denver, Colorado, April 18-27, for VS personnel from the VS Western Region. Microcomputers were used to enter simulated investigative and other field data that had been recorded on VS forms 12-27. The system is currently being upgraded to increase the speed of computerized data processing. The new computer program and supporting equipment is scheduled to be in operation by the end of the calendar year.

Secretary's Advisory Committee. The Secretary of Agriculture's Advisory Committee on Foreign Animal and Poultry Diseases met June 15-16, 1988, in Washington, D.C.,

to review current issues involving the prevention, recognition, diagnosis, control, and elimination of animal and poultry diseases foreign to the United States. The Committee made nine resolutions and recommendations and developed additional comments and suggestions related to Departmental programs on foreign animal diseases. The committee consists of representatives of the cattle, swine, poultry, and sheep industries, scientists, trade association representatives, and Government and University personnel. The Committee members are: Dr. James A. Acree, Jacksonville, Florida; Mr. Neil F. Black, Ragan, Minnesota; Mr. Clint Booth, Dallas, Texas; Mr. Ronald M. Cameron, North Little Rock, Arkansas; Mr. Dan B. Childs, Lake Placid, Florida; Dr. Walter C. Cottingham, Kingstree, South Carolina; Mr. John R. Dahl, Gackle, North Dakota; Mr. Don Gingerich, Parnell, Iowa; Dr. Frank A. Hayes, Athens, Georgia; Ms. Michelle C. Howard, Sacramento, California; Dr. John P. Kluge, Ames, Iowa; Mr. Ralph Knobel, Fairbury, Nebraska; Mr. James Nofziger, Canoge Park, California; Mr. Dean Pridgeon, Montgomery, Michigan; Mr. Jack Rundquist, Butler, Illinois; Mr. Horace Sewell, Dalton, Delaware; Mr. Ronald I. Stout, Monroe, North Carolina; Mr. Latimer H. Turner, Sarasota, Florida; Mr. Larry Werries, Springfield, Illinois; and Mr. James H. Whitmore, Springdale, Arizona. (Dr. M. A. Mixson, 301-436-8073)

Foreign Animal Disease Update

In South America, during the months of January, February, and March 1988, Brazil reported 122 herds of cattle affected with vesicular disease (foot-and-mouth disease (FMD) types O_1, A_{24}, C_3); Argentina reported 21 herds affected; Bolivia, 6 herds (FMD type A_{24}, C_3); Ecuador, 14 herds (FMD type O); and Colombia, 331 herds (FMD type O_1, A_{24}, and vesicular stomatitis). In Colombia, 136 herds were affected with the New Jersey strain of vesicular stomatitis and 36 herds with the Indiana strain.

Italy reported an outbreak of FMD type C on June 26, 1988, in a herd of 1,992 swine in the Toscany region. The control measures taken by the Government of Italy include slaughter of infected animals, quarantine of the affected geographical area, serological surveillance, and vaccination.

Israel reported outbreaks of FMD type O on June 24, 1988, in the northern districts of Zefat and Golan. By July 3, three small herds of beef cattle were affected. The control measures taken by the Government of Israel included revaccination and control of animal movements.

Kuwait reported four outbreaks of FMD type O during March 1988. The disease was initially detected on March 1, 1988. The origin of the outbreak is unknown. Properly immunized livestock were resistant to the disease. Control measures taken by the Kuwait government are: quarantine of diseased and exposed animals, closure of cattle sales markets, and vaccination of cattle, sheep, and goats in the vicinity of the outbreaks.

Other countries which reported FMD during the first 3 months of

1988 are: Federal Republic of Germany, Senegal, Chad, Oman, Sri Lanka, and Laos.

The World Reference Laboratory for FMD in Pirbright, England, reported the following FMD occurrences for the months of January, February, and March 1988: Type O Germany, Hong Kong, India, Laos, Nepal, Saudi Arabia, Sri Lanka; Type C - Philippines; and Type A - Nepal.

During February 1988, the Sri Lankan veterinary authorities reported that the **rinderpest** outbreak in their country in December 1987 was most likely introduced by goats imported from India. The diagnosis was confirmed at Pirbright and at the Indian Veterinary Research Institute, Mukteswar. Out of a total cattle population in the area of 5,067 head, 510 animals were infected, and 312 died. A total of 1,802 head of cattle and 103 goats were destroyed. Strict quarantine measures were imposed, including a ban on the slaughter of cattle and goats for consumption. Some 40,000 cattle had been vaccinated by the beginning of February 1988, around the infected area. The last case of the disease was reported on January 11, 1988. The disease is now considered to be under control.

Oman and Mali reported outbreaks of **pest des petits ruminants** during January and February, respectively.

Contagious bovine pleuropneumonia was reported in Mali in January and February and in Kuwait in March 1988.

Lumpy skin was reported in Mali, South Africa, Zaire, and Kuwait during the first quarter of 1988.

Outbreaks of **sheep pox** and **goat pox** were reported in Senegal, Mali, Tunisia, and Kuwait during the same period.

During February 1988, **African horse sickness** was reported in South Africa.

Portugal reported 10 outbreaks of **African swine fever (ASF)** in February 1988, which killed 95 of a total 1,433 exposed swine. Spain reported 31 outbreaks of ASF in February 1988.

Hog cholera was reported in the Federal Republic of Germany in February and March 1988, in the Rheinhessen Pflaz and Hannover Districts, Rhineland-Palatinate, and Lower Saxony. Five fattening pigs, 18 breeding pigs, and 60 piglets were involved. The origin of the infection is presumed to be garbage feeding. All swine on the infected farms were slaughtered and destroyed. Italy also reported an outbreak of HC in the Sardinia Region during March. Yugoslavia reported one outbreak of hog cholera (HC) in January 1988. Other countries reporting HC during the first quarter of 1988 are Malaysia, Taiwan, Mexico, Colombia, Paraguay, Chile, and Uruguay.

In India, at least two separate outbreaks of **Japanese encephalitis** (JE) occurred in the human population in late 1987.

Incomplete data indicate that approximately 100 cases (37 fatal) occurred in South Arcot District of the Tamil Nadu State and that over 100 fatalities (case total undetermined) occurred in Burdwan district, West Bengal State. Seasonal outbreaks of JE commonly occur at this time of year, and local health authorities claimed that the situation in Burdwan was less severe than in late 1986.

Namibia, South Africa, and Malaysia reported deaths in sheep due to bluetongue during the first quarter of 1988.

Epizootic hemorrhagic disease (EHD) has been reported in Canada on two occasions. The first was in 1962, when EHD virus type II was isolated from deer in the mountains of Southwestern Alberta. No other species were reported as being involved in this episode. No numbers are available on the magnitude of the outbreak. There is no law requiring the reporting of this disease to Federal authorities.

The second type II isolate was obtained in the fall of 1987 from a commercial game farm near Penticton, British Columbia in the Okanagan Valley. The deer were first ill on September 2, 1987. The disease spread to animals of other species on the affected premises and to free-ranging wild ruminants. EHD was reported later in white tail and mule deer, bison, elk, California big horn sheep, a Rocky Mountain goat, and a yak. In all, the deaths of 111 captive and free-ranging wild animals were attributed to EHD. No illness was reported in adjacent domestic sheep, goats, or cattle.

A bovine serum survey was immediately established in and around the Okanagan to determine the degree of spread of the disease. Since the clinical expression of EHD closely resembles bluetongue, sera were also examined for the presence of antibody to the latter disease. From a total of 9,224 bovines surveyed in Southwestern Alberta, Southeastern British Columbia, Okanagan Valley, and Southwestern British Columbia, 14 reacted to the serum neutralization (SN) test for EHD, and 16 reacted to the SN test for bluetongue. The reacting animals were all from the Okanagan Valley. No reactors were found in the other geographical areas.

It is believed that EHD was introduced into the Okanagan Valley by infected culicoides moving up from the United States. This belief is strengthened by reports of clinical cases of EHD-II from the States of Washington and Oregon in 1987.

It is further believed that the Okanagan Valley is the only area of Canada which, because of factors associated with vector competence, is capable of maintaining bluetongue infection. All evidence of bluetongue in Canada, both current and historical, indicates it is restricted to that valley. Because of the history of EHD, it must be recognized that the extreme southwestern part of Alberta and Southeastern British Columbia may also rarely be capable of maintaining this disease.

The United Kingdom (UK) is implementing measures to make bovine

spongiform encephalopathy (BSE) a notifiable disease. Very little is known about the cause or epidemiology of BSE, except that the symptoms and effects upon cattle are very similar to those of scrapie in sheep. BSE typically affects only one or two animals in an affected herd. A total of 511 cases of BSE have been confirmed throughout Great Britain. The UK is also banning the use of animal protein products derived from mammals in feed intended for ruminant animals, except milk and dicalcium bone phosphate. (Dr. Percy W. Hawkes, 301-436-8285)

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Central American and Caribbean Bluetongue Epidemiology

Pilot serological studies in 1980 and 1981 suggested the presence of bluetongue virus (BTV) serotypes 1, 6, 12, 14, and 17 in the Caribbean Basin. These serotypes differed from those recognized in the United States but were apprently uniform within the Caribbean Basin region. The principal insect vector of BTV in the United States, Culicoides variipennis, has not been reported in the Caribbean Basin, suggesting that BTV transmission there must depend upon some other vector species.

A regional research program was funded in 1986 through the USDA's Office of International Cooperation and Development (OICD), Special Foreign Currency Program, for BTV epidemiology in Central America and the Caribbean. Additional support is being provided through the USDA's Cooperative State Research Service and the Inter-American Institute for Cooperation on Agriculture (IICA). The program intends to provide information necessary for policy decisions on animal movements in the region.

The bluetongue epidemiology program is administered by the International Regional Organization for Animal Health (OIRSA) in collaboration with IICA, with technical support from the University of Wisconsin, University of Florida, and USDA's Arthropod-Borne Disease Research Laboratory (ABDRL). The countries participating in the study are Panama, Costa Rica, Nicaragua, El Salvador, Guatemala, Honduras, Belize, Jamaica, Barbados, Trinidad, and Tobago. Representatives of OIRSA and IICA are working in these countries with animal health officials of the ministries of agriculture to collect samples.

Twenty-five sentinel herds form the basis of the regional surveillance program. Blood samples are collected from a group of young calves or lambs in each herd at intervals of 1 to 3 months to detect BTV seroconversions. When BTV antibodies appear, the corresponding blood samples are processed in embryonated eggs and cell cultures for virus isolation. Also, insects are regularly collected in light traps located at sentinel sites. The insects are then identified as candidate vector species, their relative abundance estimated, and the possible presence of BTV determined.

The principal laboratory used for serology and virus isolation is at the Costa Rican Ministry of Agriculture and Livestock's Veterinary Diagnostic Laboratory. The laboratory operates under an agreement between the Ministry and OIRSA, with technical support from the cooperating institutions. Additional support for the identification of viruses is provided by the USDA ABDRL

laboratory.

Field work that began in 1987 is now in full swing, and data are rapidly accumulating. Preliminary data confirm the widespread distribution of BTV antibodies. Virus serotypes isolated to date coincide with those indicated in the 1980 and 1984 studies. (Dr. E. Jane Homan, University of Wisconsin, 608-262-1271)

Rift Valley Fever Update -- West Africa

Background. Rift valley fever (RVF) was recognized as a viral zoonosis more than 50 years ago in Kenya, but its real significance for human and animal health has only been appreciated in the last decade. There has been increasing awareness that the virus occurs throughout most of sub-Saharan Africa and poses a potential problem there. Epidemic RVF, with attendant abortion and death among sheep and cattle, has been recognized in Kenya, Zimbabwe, South Africa, Namibia, Tanzania, Zambia, Sudan, and Mozambique. In 1977, RVF was found outside sub-Saharan Africa for the first time: Egypt suffered the ravages of a major epidemic of animal and human disease. The extensive transmission that occurred emphasized the threat of the virus for receptive areas anywhere in the world, but the apparent disappearance of the virus from Egypt over the succeeding 3-4 years suggests that the specialized circumstances required for virus maintenance may not exist in regions outside the established endemic zone. A concise and accurate review of RVF was published in the Foreign Animal Disease Report, 10-2:9-14, September 1982.

Recent advances in RVF epidemiology. Serosurveys and virus isolation studies in most regions of sub-Saharan Africa have detected the presence of RVF virus. In the absence of epidemics in domestic livestock, infection rates in man are low, with typical neutralizing antibody rates around 1-10 percent. Disease and abortion in domestic animals also occur at low rates but are not specifically identified because of the many other livestock problems and lack of diagnostic resources in these regions. These infections are probably acquired largely from mosquitoes infected as part of the natural cycle of RVF virus maintenance. This cycle of enzootic circulation exists in many regions of Africa and may be relatively independent of epidemics, which typically center around large concentrations of sheep or cattle. In contrast to epidemics, endemic transmission occurs in both humid and seasonally arid ecosystems, and must be multifocal in nature.

There is now evidence to support the hypothesis that the mechanism responsible for these many cryptic foci may be transovarial transmission in certain Aedes mosquitoes. These mosquitoes (subgenera Neomelaniconion and Aedimorphus) oviposit in areas subject to inundation. Their eggs may remain dormant for one or more seasons until flooding triggers their development to infected adults. In Kenya, the critical species appears to be Aedes mcintoshi (formerly Ae. lineatopennis). These mosquitoes emerge when their habitats (depressions called "dambos") are flooded during particularly heavy rains. Transovarially infected larvae emerge and develop into infected

male and female adults; the females may then feed on nearby susceptible livestock. As other secondary mosquito vector populations emerge, they may be orally infected from viremic domestic animals and, if conditions are appropriate, initiate the epidemic cycle.

The potential for epizootic RVF transmission in Kenya can be predicted from satellite remote sensing. Intense rainfall leads to characteristic changes in spectral emission from local vegetation. This identifies areas of flooding sufficient to hatch the floodwater Aedes in quantities and to provide breeding sites for numerous secondary vectors, particularly Culex spp.

Human disease. RVF in man was previously thought to be a temporarily incapacitating illness with no serious sequelae. There are now reports from several countries that RVF can be accompanied by hemorrhagic fever, encephalitis, and retinal disease. These complications are uncommon; although their frequency is not known, it is estimated to be 1 to 2 percent or less of human infections.

Epidemic in Senegal River flood plain. In October 1987, physicians in the town of Rosso, Mauritania, began to see increasing numbers of patients with febrile disease and hemorrhagic fever. Rosso, a town of about 30,000 people is located about 130 km east of the point where the Senegal River empties into the Atlantic Ocean at St. Louis, Senegal. The epidemic, initially suspected of being yellow fever, was identified as RVF by scientists at the Pasteur Institute in Dakar, Senegal. During the succeeding 2 weeks, 245 cases and 28 deaths were confirmed from RVF at the Rosso hospital alone. Subsequent epidemiological investigations in man and domestic animals showed disease activity in the Senegal River flood plain from the town of Keur Massene (63 km west of Rosso), extending 225 km east of Rosso. Transmission was identified 90 km to the north along the road to Nouakchott, the capital of Mauritania.

Epidemiological studies carried out by the Pasteur Institute in more than 1,000 residents of Rosso estimated human infection rates to be about 25 percent, with 0.5 percent of infections resulting in death. Preliminary serological results suggest that the affected area may have encompassed a region with a population of 200,000 or more, leading to projected human morbidity and mortality as high as 25,000 and 1,250, respectively. Even fewer data are available to estimate the veterinary impact. In general, serological attack rates were 50 to 90 percent in sheep, cattle, and goats. Abortion rates were very high in affected areas. There was also evidence of less intense transmission extending outside the Senegal River flood plain into the river basin. This represented the first documented epidemic of RVF in West Africa and may have considerable significance for animal production and development plans in the region.

The reasons for the epidemic have not been identified. Currently, most evidence suggests that three factors are necessary for RVF epidemics to occur: (1) herds of susceptible

sheep or cattle to amplify the virus through their high serum virus titers. Although camels and buffalo may suffer disease and abortion, their viremia is lower and they probably are not the major sources of infection for arthropods; (2) dense populations of mosquitoes to transmit the virus. Other biting arthropods may be involved at times in biological or mechanical transmission; and (3) a source of virus.

Large numbers of domestic livestock belonging to local residents or nomadic shepherds were present in the area, as had been the case during other years. Thus, the availability of susceptible hosts did not seem to be the critical determinant in the Senegal River epidemic.

Residents of the area reported very high numbers of mosquitoes during the 1987 rainy season. This may have been related to the closure of the dam at Diama, 35 km from the mouth of the Senegal River. Although this dam was not intended to produce a large impoundment of water, there were instances of local flooding. Furthermore, the increased water availability in the river may have worked in concert with the ongoing irrigation projects to provide increased breeding habitats. More subtle environmental changes may also be important, such as the decrease in water salinity which the dam may have fostered in the upstream area.

The origin of the epidemic virus is unknown. There is speculation that nomadic migrations brought the virus from regions in Mauritania or Mali where previous virus activity has been documented. However, there is evidence suggesting that the virus may have originated in the Senegal River flood plain. Results of serological surveys and virus isolations have established that RVF virus has existed in many areas of both Mauritania and Senegal in the past. The activation of virus from an established focus would also be consistent with today's concepts of RVF virus ecology. This is further supported by the finding of simultaneous virus activity in The Gambia.

Transmission in The Gambia. Investigations in The Gambia during January 1988 showed serological evidence of RVF virus activity. There was the usual high level of febrile disease and livestock abortions during the rainy season, but no clinical diagnosis of epidemic disease. Nevertheless, about 22 percent of 132 livestock had RVF-specific antibodies, and 7 percent of these were also IgM positive, suggesting recent transmission. The level of transmission was much lower than seen in a typical study area in the Senegal River flood plain, where 72 percent had IgG antibodies and 76 percent of these also had RVF-specific IgM. This finding has several interpretations, but the two most important parallel the possibilities alluded to in the Senegal situation: (1) the virus was introduced from the epidemic to the north and either did not have time or appropriate conditions to reach epidemic levels in The Gambia before the dry season, or (2) the virus existed in The Gambia and 1987 was a year of moderate transmission. The latter hypothesis is supported by retrospective serological surveys that detect low but significant prevalence of RVF antibodies in human sera collected in The

Gambia in 1982 and several subsequent years.

Advances in diagnosis. Classical techniques of virus isolation and serology on paired serum samples have been successful in RVF diagnosis in the past. The Senegal River epidemic produced an opportunity to examine recently developed diagnostic methodologies. ELISA techniques can detect antigen in serum and aborted material of ewes infected in the laboratory. Antigen was found in 30 percent of the viremic human samples from this epidemic. Although not as sensitive as virus isolation, antigen detection by ELISA was rapid, specific, and would reliably diagnose epidemic transmission.

IgM antibody ELISA techniques provided the greatest practical advance in diagnosis. Acute cases in man and animals could be diagnosed with a single serum sample even after the cessation of viremia. It was also possible to survey areas for evidence of recent transmission comparing IgM and IgG RVF antibodies. "IgM capture" format seemed to be important in the sensitivity and specificity of the results.

Hybridization with radioactive viral nucleic acids has been used to detect RVF virus in laboratory samples, including tissues from infected sheep fetuses. This technique was useful but less sensitive than ELISA antigen detection in human sera from the epidemic. Future advances may increase sensitivity and develop a nonradioactive format.

The utility of these tests is further enhanced with a biohazardous agent like RVF virus. They can be performed with inactivated reagents on inactivated samples.

Another diagnostic aspect which is under development may also have field utility. Formalin-fixed tissues in the laboratory have been useful substrates for enzyme immunochemistry and nucleic acid hybridization.

Vaccines. Effective veterinary vaccines for RVF are in use in several countries of sub-Saharan Africa. The live-attenuated Smithburn strain provides long-lasting immunity, but is pathogenic for the fetus of pregnant ewes. A formalin-inactivated product is not dangerous for pregnant ewes but provides only short-term immunity and requires high manufacturing standards to assure the absence of residual live virus.

Newer vaccines are under development, including an intensively mutagenized live-attenuated strain. Initial studies of this virus have shown safety for lambs and pregnant ewes and a lack of reversion. More extensive field studies are expected in 1988 and 1989.

The primary approach for protecting man from RVF is control of livestock infection. Current evidence suggests that limiting amplification of virus in domestic animals will block extensive human disease. An inactivated RVF vaccine has been effective in

preventing disease in laboratory workers. This vaccine was produced by a technology similar to that used for the Salk polio vaccine. It has been used in several thousand people and appears to be safe, although sufficient experience has not been accumulated to permit licensing in the United States. This vaccine may be indicated for persons who are at particular risk, such as abbatoir workers or veterinarians.

The wide distribution of viral antibodies throughout sub-Saharan Africa suggest that RVF may have a significant public health impact on the continent, but their low prevalence does not justify the large-scale use of an investigational vaccine that is expensive and in limited supply.

Implications for development in Africa. The 1987 epidemic in the Senegal River flood plain shows once again that RVF epidemics can occur in unexpected regions and emphasizes the importance of the very widespread enzootic distribution of the virus throughout sub-Saharan Africa. The relation of this epidemic to the water management efforts of the Senegal River has not been defined, but certainly RVF must now be a major consideration in future planning of such projects. In east Africa, new concepts of the RVF endemic cycle and determinants of epidemic disease have led to predictive models for epidemics and innovative strategies that could be applied to control emergence of floodwater Aedes mosquitos. Our knowledge of RVF viral ecology elsewhere in Africa lags far behind. New and effective diagnostic tools should allow us to identify RVF more rapidly and to better define the contribution it makes to human and animal disease in Africa. Improved livestock vaccines are under development and could be much more effectively deployed if remote sensing and other ecological predictors can be refined. (~~Col~~. C. J. Peters, M.D., USAMRIID, Fort Detrick, Maryland 21701-5011, 301-663-7193)

Focus on... Rinderpest

Rinderpest (RP) is a contagious viral disease primarily affecting cattle and domestic buffaloes. The disease appears in a spectrum of clinical forms varying from severe disease with high morbidity and mortality in highly susceptible stock in epidemic areas to mild, almost subclinical infection in resistant stock in endemic areas. The epidemic form of RP is probably the most devastating disease seen in cattle in the world, reflected by the English and French synonyms, cattle plague and la peste bovine.

RP History

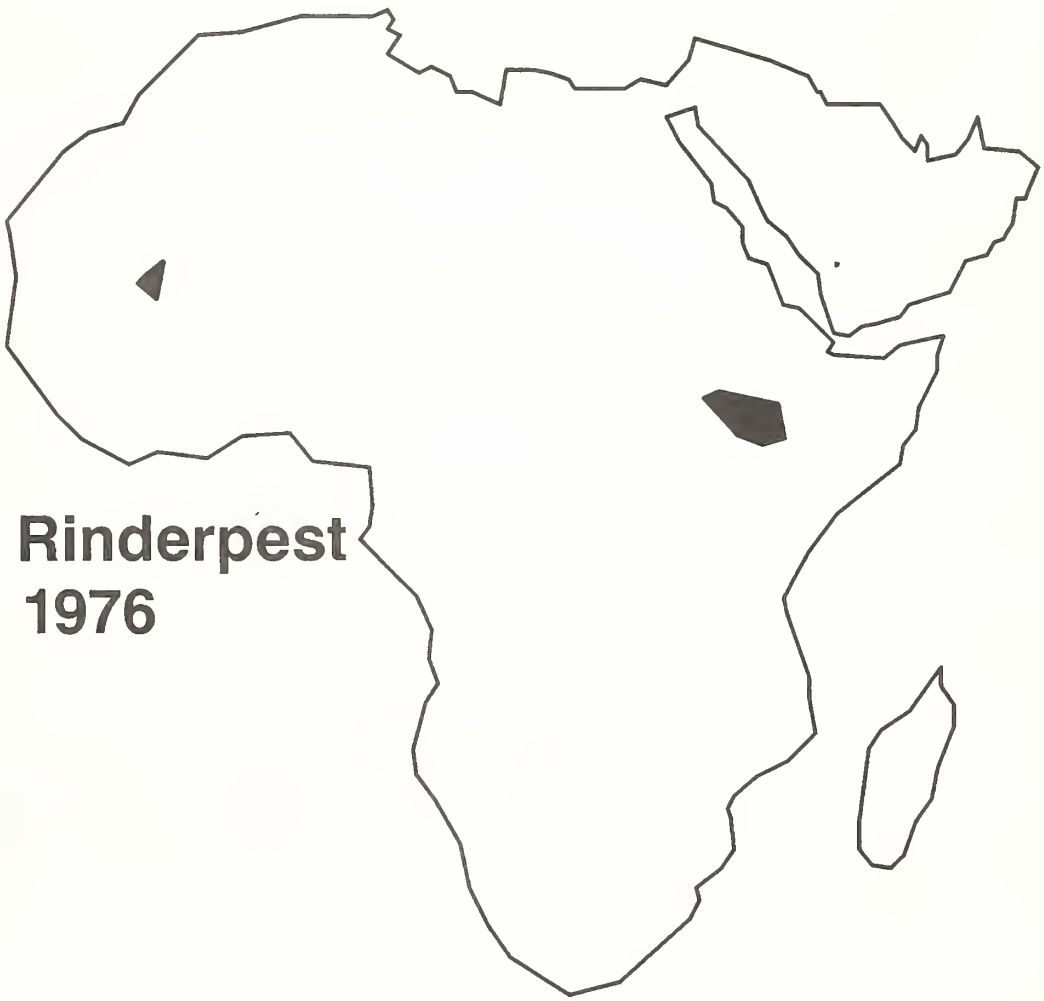
RP probably originated in Asia and periodically entered the Middle East and Europe with invading armies from the east, causing widespread destruction of cattle. The economic losses caused by these epidemics inspired the establishment of veterinary schools, first in France and then throughout Europe.

The disease was eradicated from most of Europe toward the end of the 19th century by slaughter and movement control. In 1889, RP was reintroduced into Africa in Somaliland from whence, as "The Great African Pandemic," it spread westward to reach Senegal and

southward, reaching the Cape by 1897. The loss of many millions of cattle and innumerable susceptible wildlife destroyed the economy of pastoral tribes, financially ruined ranchers and farmers and may have even altered the continental ecology.

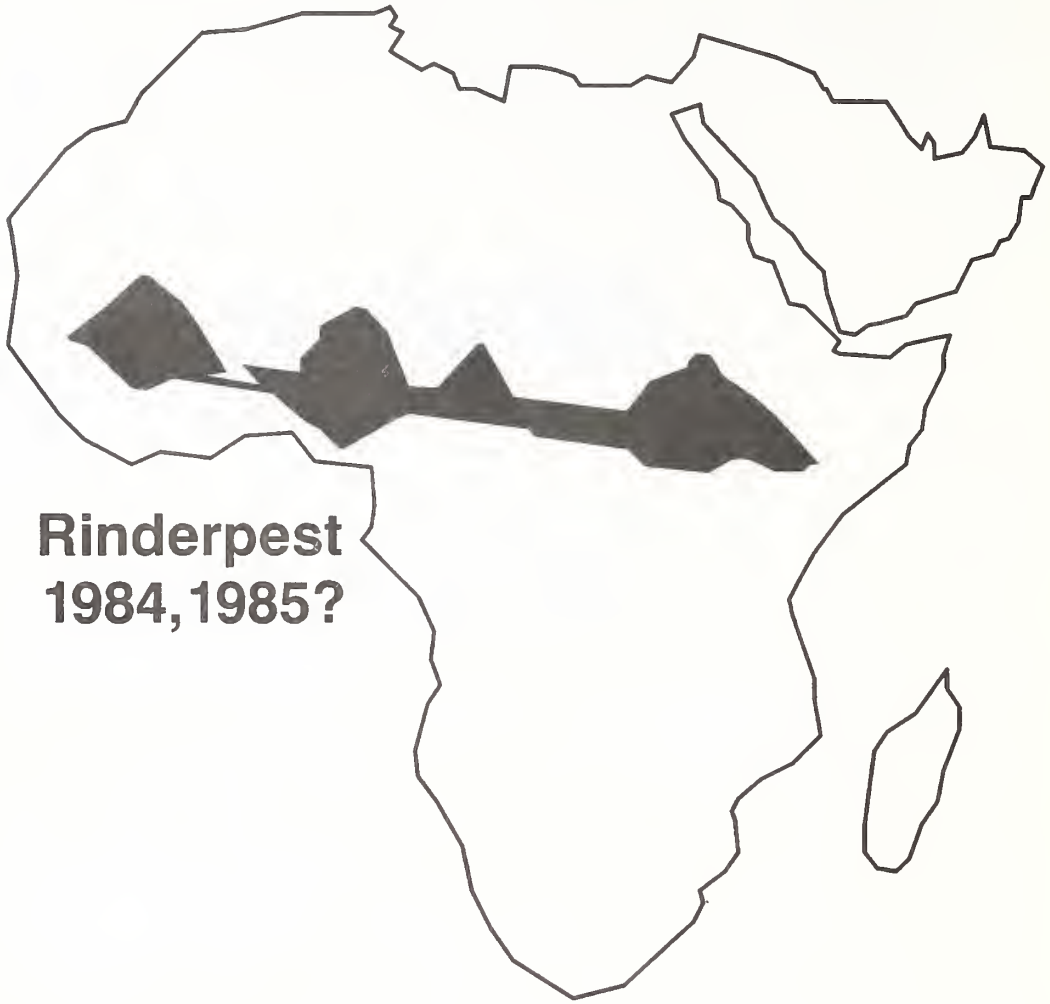
The disease persists in equatorial Africa, the Indian subcontinent and the Middle East. The threat of RP to other areas is best illustrated by one of the last European outbreaks of the disease in Belgium. The only reported RP outbreak in the western hemisphere, in Brazil in 1921, was caused by the shipment of infected cattle from India in 1920.

Between 1962 and 1976, a joint project of the Organization for African Unity (OAU) and donor organizations, known as project JP15, tried to control and possibly eradicate RP from Africa through vaccination. Project JP15 dramatically reduced the continental incidence of the disease to a handful of outbreaks in two endemic foci.


Rinderpest
1976

One of these outbreaks was in the northern part of east Africa. The other was in the Sahelian region of west Africa. The end of project JP15 vaccination in 1976 coincided with a worldwide economic recesssion. The expense of maintaining annual vaccination campaigns became too high for many African countries. Subsequently, RP emerged from its two strongholds, and a new pandemic affected cattle throughout west, central, and parts of east Africa between 1979 and 1983.

The situation was rapidly brought under control through internationally financed mass vaccination campaigns which were efficiently carried out by national veterinary authorities.



RP Persistence

RP persists at low incidence in areas where disease control measures have been neglected, in eastern Uganda, southern Sudan, Chad, and Ethiopia.

RP flared up in the Near East in Lebanon, Syria, and Israel during the early and mid-80's, following civil and military disturbances there. Vaccination controlled these outbreaks. Isolated outbreaks have occurred in countries on the Arabian peninsula following the importation of contaminated cattle and buffaloes intended for slaughter. RP persisted in India throughout the 1980's despite massive vaccination programs and was reported in Sri Lanka in October 1987.

Susceptible Species

All cloven-hoofed animals are susceptible to infection by RP virus, but there is a gradation in the degree to which each species suffers from clinical disease. This may vary with different strains of the virus. Among domestic stock, cattle

and buffaloes are highly susceptible while sheep and goats are much less so. Asian domestic swine are highly susceptible, but European breeds suffer only subclinical infections. Camels develop subclinical infections following experimental inoculation.

Many wild animals are highly susceptible to severe clinical disease. Eland, kudu, buffalo, giraffe, and warthogs are particularly at risk in Africa. Nilgai, deer, and buffalo are at risk in Asia.

In Africa, RP is largely confined to cattle with occasional subclinical infections in sheep and goats and clinical outbreaks in susceptible wildlife. Sicknesses resembling RP in African sheep and goats are nearly always due to the virus of pest des petits ruminants (PPR). In India and parts of the Middle East where PPR is not found, RP affects cattle, sheep, goats, and pigs.

RP Virus

RP virus is a member of the genus Morbillivirus, in the family Paramyxoviridae, and is therefore a single stranded RNA, enveloped virus. The infective particle is pleomorphic in shape, approximately 100-300 nm in diameter and contains a coiled filamentous or tubular nucleocapsid. RP virus has been experimentally adapted to grow in rabbits, hamsters, and chicken embryos. The virus is readily inactivated by fat solvents, betapropiolactone, ultraviolet light, heat and high or low pH. Glycerol is deleterious to RP virus and therefore should not be used as a preservative. Putrefaction of contaminated carcasses rapidly inactivates the virus.

Transmission

RP is maintained by a simple direct cycle of infection from an affected animal to a susceptible one. The virus is excreted in most bodily excretions, especially oculo-nasal discharges and feces, from 1 to 2 days before the onset of clinical disease until 8 to 9 days after clinical disease starts.

Indirect contact plays only a limited role in RP transmission, and airborne transfer probably only occurs across short distances in confined spaces. The route of infection is probably via the naso-pharynx and upper respiratory tract.

In epidemic areas, RP spreads rapidly throughout the population at risk. In endemic areas, RP is maintained by the introduction of susceptible animals and is therefore a disease of younger animals in which maternal immunity has waned some 3 to 9 months after birth. Biting arthropods and vertical transmission are not involved in the maintenance of RP.

Pathogenesis

The incubation period varies from 3 to 4 days following parenteral inoculation, 7 to 10 days following experimental contact exposure, and longer under natural conditions. Infection via the upper respiratory tract leads to virus multiplication in draining lymph nodes, followed by generalization to other lymphoid tissues where the virus grows readily to high titers. Viremia ensues with virus dissemination to and growth in the epithelial tissues,

especially those of the alimentary tract. The virus causes necrosis and, in severe cases, the consequent loss of epithelium diarrhea leads to dehydration and death.

Clinical Signs

Fever (up to 40°C or 104°F) is the first clinical sign followed 2 days later by erosive stomatitis, oculo-nasal discharge depression and anorexia. Diarrhea begins 3 to 5 days after the onset of fever and persists for up to a week. The diarrhea may contain blood, mucus, and epithelium. Severely affected animals are unable to stand and show signs of abdominal pain, rapidly become dehydrated, and may die 6 to 12 days after the onset of illness. Some affected animals linger for 2 to 3 weeks. Those that recover have prolonged convalescence. Pregnant survivors may abort about 2 to 3 weeks after the onset of illness.

Milder cases of RP, which are the more common form in endemic areas, may demonstrate all of the main symptoms to a lesser degree or may only show fever and transient mouth lesions.

Post Mortem Findings

The coat is soiled, rough and dull, and the body condition is poor and usually dehydrated. Lymphoid tissues are slightly enlarged, edematous and may be congested or occasionally haemorrhagic. Congestion and erosions are found throughout the alimentary tract. Initially the lesions are pinhead in size. These may then enlarge and coalesce. Peyer's patches are necrotic. Congestion of the crests of the longitudinal folds of the colon and rectum produces classic "zebra striping." There may be congestion and erosion in the upper respiratory tract and the female lower genital tract.

Diagnosis

Although a presumptive diagnosis may be made based upon clinical and pathological changes, the disease must be confirmed in the laboratory, especially when it occurs in new geographic locations. Laboratory diagnosis is done by recovering and identifying infectious virus or by demonstrating specific RP viral antigens in the tissues of affected animals. Virus and viral antigen titers are highest during the first 4 to 6 days after the onset of fever but may be detected at lower levels just before the onset of fever and for up to 10 to 12 days after the onset of fever.

Samples should be collected from animals that have fever and mouth lesions at a stage of the disease when they may have only started to show diarrhea. Samples collected from animals which have had diarrhea for several days may be negative. The virus may be isolated from eye swabs, gum swabs, blood leucocytes or lymph node biopsies. Rapid antigen detection by micro-immunodiffusion or counter-immunoelectrophoresis (CIEP) can be made using eye swabs, gum swabs, or lymph node biopsies as sample material. Samples of spleen, hemolymph node, peripheral and gut lymph nodes, and gut lesions should also be tested. Where possible, samples should be collected from at least six affected animals. Samples should be placed on ice, but not frozen, and transported to a diagnostic laboratory as rapidly as possible. Glycerol destroys RP virus.

Paired serum samples collected from animals with RP ordinarily show rising antibody titers. Neutralizing antibodies develop 5 to 6 days after infection, rise rapidly to titers greater than 2.0 $\log_{10}$ VN_{50} (virus neutralization 50 percent end point), decline slowly, and usually remain at detectable levels for the rest of the animal's life. The enzyme-linked immunobsorbent assay (ELISA) test can detect persistent antibodies and, therefore, may be useful for epidemiological surveillance. Several other serological tests such as the complement fixation test, CIEP test, and indirect immunofluorescence test (IIF) tend only to detect relatively short-lived antibodies.

Differential Diagnosis

Severe RP is clinically and pathology indistinguishable from the mucosal disease form of bovine virus diarrhea, although the percentage mortality is usually much higher in RP. Other diseases which occasionally resemble RP are infectious bovine rhinotracheitis, malignant catarrhal fever, and Jembrana disease. (See 12-4:3-6 for malignant catarrhal fever review and 13-3:10-13 for Jembrana disease review.) Milder forms of RP especially in younger animals must be differentiated from conditions causing erosions or ulcerations of the mouth.

Control

Swift diagnosis must be followed by strict quarantine to control RP. Slaughter and safe disposal of infected animals is effective for control when this practice is included under official disease control policy. Since the disease readily spreads by close contact, movement restriction of all susceptible animals is important. In endemic and "at risk" areas, annual vaccination is recommended, using the excellent attenuated cell-culture vaccine now widely available to confer lifelong immunity against all strains of the virus. It is essential that a high level of herd immunity be established if eradication is the aim, otherwise mild strains of the virus may persist. In epidemic or virgin areas, vaccination should be avoided until absolutely necessary since serological tests cannot distinguish between antibody induced by vaccine and field strains. The presence of vaccinal antibodies complicate serological surveillance. RP can cause severe but self-limiting disease outbreaks in susceptible populations of wildlife. The large populations of susceptible wildlife in east Africa do not appear to maintain RP virus indefinitely, although there is evidence that the virus may persist in them for more than a year.

Control and Eradication Programs

Recent RP epidemics in Africa encouraged a renewed drive to eradicate RP from the continent. The Pan African RP Campaign (PARC) started in 1987. It is financed by the European Economic Community, advised by specialist agencies including the United Nations Food and Agriculture Organization (FAO) and United Kingdom Overseas Development Agency (ODA), and implemented through the OAU. The primary aim of PARC is to control RP through strategic aid, where necessary, and to improve veterinary services in Africa. If good control of RP is achieved, then PARC will push for complete eradication. Similar programs have been discussed internationally for the

eradication of RP in the Middle East and in southern Asia.

Research Needed

Surveillance should be maintained in countries with endemic RP to confirm the continued presence of RP virus in asymptomatic sheep, goats, and wildlife. These species comprise useful sentinel populations because antibodies in the young give a warning of the recent circulation of RP virus in nearby cattle.

Monoclonal antibodies and molecular virological techniques should be developed to distinguish between different strains of the virus. For sero-epidemiology, a test is needed to distinguish between vaccine-induced antibodies and field virus-induced antibodies in cattle. An understanding is needed of the mechanism by which RP virus changes virulence from predominantly virulent strains in epidemics to predominantly mild strains in endemic areas.

With respect to diagnosis, a number of reliable tests are available. Therefore, emphasis should be placed on increasing the number of samples submitted for diagnosis. Research is needed to increase vaccine thermostability and half-life in the field. It would be difficult to find a better vaccine than the one now in use. (P. Rossiter, Overseas Development Agency of the United Kingdom, Kenya, Africa, and C. M. Groocock, USDA, APHIS, Box 351, APO New York 09675)

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